CASE REPORTS

Cortisone, Corticotropin and Procaine in the Treatment of Corrosive Esophagitis

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THE ACCIDENTAL or intentional ingestion of corrosive chemical agents frequently causes scarring and incapacitating stricture of the esophagus.

The site of stenosis is determined by development of segmental spasm, usually localized in one segment or another of the organ. The effects are inconsequential if only the epithelium is destroyed, but they may be severe if the submucosa or the muscular layer is involved, for the resultant healing by granulation tissue leads to progressive narrowing of the lumen of the esophagus by stricture formation, which usually becomes manifest in from one to three months after injury.

Measures to dilute and neutralize swallowed corrosive chemicals are of first importance in early therapy, and administration of local anesthetic agents may help diminish intense dysphagia.[3] Efforts directed toward retarding and lessening the development of cicatrization by use of antibiotics and steroid hormones are in order in light of good results reported in the treatment of experimental corrosive esophagitis in rabbits when these agents were used.[2] It has been demonstrated that the inhibiting effect of cortisone on granulation tissue does not interfere with the process of epithelization.[1, 4]

In a previously reported case, benefit was noted from administration of corticotropin (ACTH) and procaine by mouth early in the course of esophagitis caused by the ingestion of Lysol.[3] Herein is another case in which cortisone was given intramuscularly and procaine by mouth to a patient with corrosive esophagitis due to ingestion of lye crystals. At autopsy later there was evidence that the therapy was effective in preventing the usual late cicatricial sequelae.

REPORTS OF CASES

A 74-year-old man was admitted to the Long Beach Veterans Administration Hospital four hours after having ingested an unknown quantity of lye crystals in an attempt to kill himself. He had vomited bloody material soon after swallowing the lye and some two hours later had been given vinegar to drink, according to a son, who said also that the patient had been deteriorating mentally for several years and thrice before had attempted suicide.

At the time of admittance the patient was acutely ill, confused, disoriented and vomiting. He was unable to speak above a whisper and complained of difficulty in swallowing. The blood pressure was 150/80, the pulse rate 100 and the temperature 99.2° F. The lips and tongue and the palatal, buccal and pharyngeal mucosa were swollen, reddened and ulcerated. There was diffuse tenderness in the epigastrium.

A total of 16 ounces of half strength vinegar solution was administered orally during the first two hours of hospitalization. Penicillin, 300,000 units, in combination with Streptomycin, 0.5 gm., was given parenterally and continued at 12 hourly intervals. Parenteral hydration was carried out for three days and no food was given by mouth. Administration of 1 per cent procaine solution in doses of 15 cc. every two hours and 100 mg. of cortisone daily, given intramuscularly in divided doses, was begun on the second day. Procaine therapy was continued for one week and cortisone for 15 days.

Restraint of the patient was necessary, owing to confusion, restlessness, delusions and disorientation. These symptoms were present before cortisone administration was started and, except for occasional short periods during which the patient was oriented and cooperative and would exclaim, "What a fool I was," they continued throughout the period of hospitalization. As the patient did not urinate, a catheter was kept in place.

The oral and pharyngeal edema, ulceration and inflammation increased during the first three days, then gradually subsided. Epithelization of the lesions and complete healing took approximately 20 days. By the eighth hospital day the patient was swallowing liquids well but refused to eat and was fed by tube. During the first few days of tube feeding, creamy purulent exudate was present in the gastric aspirate, at first in moderate amounts and later in

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diminishing quantity. Fever, present generally but intermittently throughout the period of hospitalization, was attributed to bronchopneumonia and pyelonephritis. The penicillin-streptomycin combination originally instituted was changed to aureomycin, 500 mg. every six hours; and later, because of onset of diarrhea, terramycin, 250 mg. every six hours, was substituted. No recurrence of fever was noted after the twentieth hospital day. Upon advice of a psychiatrist that the patient was still suicidal and acutely psychotic, he was committed to a state mental hospital, where he died three weeks later. No pathologic change was noted in careful postmortem examination of the mouth, larynx, pharynx, esophagus and stomach. The mediastinum and pleural cavities were normal. The pathologic diagnosis was bilateral bronchopneumonia and cerebral arteriosclerosis with cortical atrophy.

(The previously mentioned and previously reported[3] case of damage to the esophagus by Lysol was that of a man 40 years of age who was admitted to the Long Beach Veterans Administration Hospital six hours after the ingestion of two ounces of the caustic material in a suicidal attempt. Vomiting ensued and emergency treatment consisted of gastric aspiration. The body temperature was 101° F. at the time of admittance. The tongue and buccal mucosa were inflamed and there was a grayish membrane in the oropharynx. Corticotropin (ACTH) was given intravenously, 10 mg. daily in divided doses over a period of 18 hours with aureomycin 1.0 gm. daily for 17 days. On the third hospital day the patient was given 15 cc. of 1 per cent procaine solution to gargle and swallow after each meal. The following day a bland diet preceded by procaine swallows was well tolerated. Esophagram was normal one week following completion of therapy and the patient was followed for five months following discharge during which time he continued well.)

DISCUSSION

In neither of two cases in which therapy with cortisone or corticotropin and procaine by mouth was given to patients who had swallowed strong corrosive chemicals was there evidence of esophageal stricture. Of course there is no way of knowing in any given case whether or not esophageal strictures will result from the swallowing of corrosive chemicals. Moreover, it is possible that concentrated solutions may, upon contact with the lining of the oral and pharyngeal cavities, cause such powerful spasms of the pharyngeal muscles as to prevent the swallowing of the chemical. However, it was very strongly indicated by the symptoms — hematemesis, epigastric tenderness, severe dysphagia, purulent exudate in the stomach—that esophagitis did occur in one case at least and that the esophageal changes were similar to those noted in the buccal and pharyngeal mucosa. It may be conjectured that the administration of the steroid hormones, coupled with the use of antibiotics to reduce secondary infection, was responsible for sufficiently suppressing granulation tissue formation to avert the formation of stricture in these two cases. In any event, these results are sufficiently encouraging to warrant further trial of this form of therapy in cases of corrosive esophagitis.

SUMMARY

Two suicidal patients suffering from the corrosive effects resulting from the ingestion of strong chemicals were treated with cortisone or corticotropin, antibiotics and procaine solution. Treatment was begun early in the course of the disease. Esophageal stricture did not result.

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Anaphylactic Reaction to Penicillin O

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IMMEDIATE ANAPHYLACTIC REACTIONS to penicillin G have been reported with increasing frequency. In several cases such reactions have caused death. It is likely that such untoward accidents are more common than published reports would indicate. The literature on the subject of anaphylaxis to penicillin was recently adequately reviewed by Feinberg, Feinberg and Moran.[1] In most instances a history of allergic disease was obtainable, penicillin had been given previously, often followed by untoward reaction, and the immediate reaction to intradermal test with dilute penicillin solution was positive.

Allylmercaptomethyl penicillin (penicillin O) has been described as "anti-allergic." Patients in whom allergic phenomena developed after administration of penicillin G, later received penicillin O without ill effect.[2, 3] While this observation may be generally true, one of the authors (H.B.) has observed unequivocal evidence of cross sensitivity of the delayed type. Feinberg and co-workers[1] demonstrated cross-antigenicity between penicillin O and penicillin G and predicted that penicillin O might be anaphylactogenic. The following case report is submitted to warn physicians against relying implicitly on the "anti-allergic" properties of penicillin O in the management of patients sensitive to penicillin G.

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